AH and 39 (23%) had masked AH. Two-hundred eighteen subjects (86%) were treated with continuous positive airway pressure. Masked AH was not associated with the severity of OSAS (AHI and the percentage of total sleep time during which the oxygen saturation was <90% (SaO_2<90%). However, subjects with masked arterial hypertension have a worst metabolic risk profile compared to normotensive patients.

Conclusions. Masked hypertension is frequent in OSAS patients and is not associated with the severity of the OSAS.

Table – Results

	Masked AH n=39	No AH n=80	p
Apnea-Hypopnea Index	48.9±23.8	47.7±24.4	0.810
SaO_2 <90%,%	12.0±15.3	14.0±17.7	0.579
Age, years	49.6±6.8	49.6±9.4	0.995
Body mass index, kg/m²	30.1±5.5	30.0±5.4	0.947
Fasting glycemia, mg/dL	94±13	94±34	0.148
Homeostasis model assessment-estimated insulin resistance (HOMA-IR)	3.9±2.9	2.5±2.8	0.031
Fasting triglyceridemia, mg/dL	170±110	130±90	0.004
CRP us, mg/L	4.9±6.6	3.8±2.5	0.980
Metabolic syndrome, n (%) NCEP ATP III definition	13 (33)	10 (13)	0.007

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Peripheral arterial disease remains underdiagnosed even in post hospital discharge for any ischemic event

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Objective: To evaluate the prevalence rates of peripheral arterial disease (PAD) diagnosed before and during the general practitioner (GP) consultation following hospitalization for ischemic event.

Method: In a cross sectional study, 102 Belgian GPs recruited 505 patients of ≥50 years, hospitalized within the last year for any ischemic event. The Screening CardioVascular Lab (SCVL®, GenNov) automated combination of the oscillometric determination of the ankle-brachial index (ABI) and of the outcome of the Edinburgh questionnaire (EQ). This device integrated the case report form.

Results: The patients were 69±11 years old; men were 63%. Their other CV risk factors were: hypertension in 75%; dyslipidemia in 74%; sedentarily in 53%; abdominal obesity in 52%; familial history of CVD in 38%; smoking in 28%; diabetes in 22%. The arterial events, which induced the recent hospitalizations, were: acute in 71% of cases, planned for revascularization in 23%; coronary in 50%, cerebral in 29%, aortic/peripheral in 15%. For at least their hospitalization, 95% of patients were treated with antithrombotics, including 88% with antiplatelets. At the GP visit, PAD was unknown in 10% of cases and previously diagnosed in 19%, including an appraisal exclusively on clinical signs in 5% and a confirmation by Doppler ultrasonography or arteriography in 13%. The EQ had a positive outcome in 16% of patients: the site of claudication was typical in 85% of these; its severity, of grade 2 in 52%. The patient ABI was of 0.95±0.20, <0.90 in 37% of patients and >1.30, in 3%. The combination EQ~ABI was abnormal in 46%.

Conclusion: In patients recently hospitalized for any ischemic event, the "true" prevalence rate of PAD, as diagnosed prior to the GP visit, was 19%. The "apparent" one, by using the SCVL® combining the ABI determination and the Edinburgh questionnaire at the GP office, was 46%, despite the extensive antithrombotic treatment. Even in a hospital setting, PAD is widely underdiagnosed.

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Arterial hypertension profile is associated with both the severity of OSAS and metabolic syndrome

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Background: Obstructive sleep apnea syndrome (OSAS) is associated with an increased risk of arterial hypertension (AH) and cardiovascular complications. The aim of this study was to evaluate whether the AH profile, already known or newly diagnosed, was associated with the severity of the OSAS.

Methods: Clinical blood pressure and twenty-four-hour ambulatory blood pressure monitoring were systematically recorded in 253 consecutive patients with documented OSAS.

Results. In this cohort (mean age 51.3±9.5 years, 83% men, mean BMI 31.8±5.8 kg/m², mean Apnea-Hypopnea Index (AHI) 52.9±28.5%), 82 (32%) had known AH, 91(36%) had newly diagnosed AH and 80 (32%) had no AH including white coat effect (25 [15%]). Two hundred and nineteen patients (86%) were treated with continuous positive airway pressure with no difference with regard to the AH profile (p=0.305 for trend). The AHI was higher in OSAS hypertensive patients compared to patients without AH (Table) associated with an increased rate of the metabolic syndrome and worst insulin resistance profile.

Conclusions: We found a continuous relation between the severity of OSAS, the presence of AH whether already known or newly diagnosed and the metabolic profile in a large cohort of patients with treated OSAS.

Table – Results

	Known AH n=82	Newly diagnosed AH n= 91	No AH n=80	p
Apnea-Hypopnea Index	56.8±33.5	53.4±25.9	47.7±24.4	0.015
SaO_2 <90%,%	19.0±22.0	18.0±20.0	14.0±17.7	0.158
Age, years	55.1±7.9	50.0±8.6	49.6±9.4	0.307
Body mass index, kg/m²	33.9±6.2	31.4±5.3	30.0±5.4	0.299
Fasting glycemia, mg/dL	106±29	97±16	94±34	<0.001
Homeostasis model assessment-estimated insulin resistance (HOMA-IR)	4.3±3.30	3.7±3.1	2.6 ±2.5	0.06
Fasting tryglyceridemia, mg/dL	170±150	158±94	130±90	<0.001
CRP us, mg/L	3.8±2.5	4.1±4.7	3.85±3.7	0.54
Metabolic syndrome, n (%) NCEP ATP III definition	60 (73.2)	45 (50)	10 (12.5)	<0.001

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Efficiency of abdominal CT scan as first line investigation in resistant hypertensive patients with adrenal cause suspicion

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Objective: To evaluate the efficiency of adrenal imaging by an abdominal CT scan as first line investigation in subjects with resistant hypertension when

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an adrenal cause is suspected although hormonal tests are recommended as first line.

Methods: On 134 hypertensive patients uncontrolled by at least a combination therapy and with a suspicion of secondary hypertension due to adrenal cause, an abdominal CT scan was performed in first intention. In all subjects, an exploration of the renin-aldosteron axis in standardized conditions, a 24-hour urinary cortisol and a WHO recommended biological analysis were also performed afterwards.

Results: An abnormal morphology of adrenal was detected in 57.5% of patients. Observed abnormalities were: bilateral hyperplasia (30.6%), unilateral adenoma (15.7%), unilateral hyperplasia (6.7%) and bilateral adenoma (4.5%). Abnormal adrenal hormonal tests were recorded in 16.4% of patients, with a primary aldosteronism (A/R corrected> 23) found in 10.4% and increased urinary cortisol in 6%. Treatment implementation was decided upon morphologic and hormonal results. At 6 months of follow-up, a controlled blood pressure was observed in 73.1% of subjects, spironolactone had been prescribed in 41.8% of subjects and adrenal surgery performed in 3.7% of patients. If hormonal tests had only been performed, a retrospective analysis showed that treatment with spironolactone would have been prescribed only in 12.6% of subjects and an adrenal surgery been proposed only in 2.9% of patients.

Conclusion: In our population of patients with a resistant hypertension and adrenal disease suspicion, abdominal CT scan as a first line investigation leads directly to the conclusion of an adrenal aetiology in 45.5% of patients. In contrast, a specific treatment would have been undertaken only in 15.5% of patients if screened by hormonal examinations only. This study suggests that in subjects with uncontrolled hypertension and suspected to have an adrenal cause, abdominal CT scan is an efficient first line investigation.

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Do vascular markers predict cardiovascular death in primary prevention?

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Purpose: The purpose of the study was to determine the prognostic significance of vascular markers in apparently healthy subjects.

Methods: Analyses were based on the Third South-Western French MONICA Survey (1995-1996) carried out in participants aged 35-64. Vital status, date and cause of death were obtained 14 years after inclusion. Subjects with a personal history of Atherosclerotic Cardiovascular Disease or with a personal history of Severe Chronic Disease were excluded. Pulse Wave Velocity (PWV), Pulse Pressure (PP), presence of carotid or femoral atherosclerotic plaques, and carotid Intima-Media Thickness (IMT) were assessed. Identification of the determinants of cardiovascular mortality was based on a multivariable survival analysis.

Results: There were 1144 participants (587 (51%) men). Over the 14-year study period, there were 63 deaths, 21% due to a cardiovascular cause (Ischemic Heart Disease, Atherosclerotic Cerebrovascular Disease or Atherosclerosis). The median 10-year risk of coronary event according to the Framingham Risk Score (FRS) was 6.4%. PWV, PP, and carotid or femoral atherosclerotic plaques were all significant and independent determinants of cardiovascular mortality, whereas carotid IMT was no longer associated with cardiovascular mortality after adjustment for FRS. The addition of PWV, PP or atherosclerotic plaques to FRS in a prediction model resulted in an improvement of the discriminatory value of the model as shown by the C-statistic which was 0.76 [95% CI: 0.61-0.90] for FRS alone, 0.80 [95% CI: 0.64-0.95] for FRS and PWV, 0.80 [95% CI: 0.65-0.95] for FRS and PP, and 0.77 [95% CI: 0.60-0.94] for FRS and plaques. Using PWV, PP and plaques in the prediction model led to a Net Reclassification Improvement of 22, 19 and 25%, respectively.

Conclusion: Vascular markers are independent determinants of cardiovascular mortality risk in apparently healthy subjects in primary cardiovascular prevention and improve classification of subjects.

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Long term prognosis value of ankle brachial index in coronary artery disease

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Purpose: Our objective was to assess the long term prognosis value of ABI in a contemporary large cohort of pts with known CAD.

Methods. Among 834 consecutive male pts hospitalized in 2001-2004 for coronary artery disease, ABI was measured in 776 pts. ABI –0.9 was considered as abnormal. ABI –0.9 and > 0.6 was considered as low and ABI≤0.6 as very low. The median follow-up was 7.17 years. Total mortality was predicted with a Cox proportional hazard model.

Results: Mean age (SD) was 60.2 (8.1), 144 pts (18.4%) were diabetic, 155 pts (19.8%) were smokers, and median heart rate was 61 bpm [Interquatile range (IQR)] [57-70]. Mean left ventricular ejection fraction was 0.53 (0.13). 88.5% were on antiplatelet therapy, 75.2% on beta-blocker, 66% on statin therapy and 54.8% on ACE inhibitors or ARB.

The sample comprised 518 pts (67%) with normal ABI and 258 (33%) with abnormal ABI (215 (83.3%) pts had a low and 43 (16.7%) a very low ABI). The cumulative seven-year total mortality rate was 17.6%. In the normal ABI group, mortality rate was 13.9%, whereas it was 21.4% in the low and 44.2% in the very low ABI group (p<0.001).

After multivariate adjustment for age, diabetes, tobacco consumption (none; ≤40 pack-years; >40 pack-years), heart rate, duration of CAD, left ventricular ejection fraction (>0.5; ≤0.5 and > 0.35; ≤0.35), history of chronic obstructive pulmonary disease or stroke, statin therapy and coronary revascularization, hazard ratio (HR) for all-cause death was 1.36 (95% CI [0.92; 2.03] p=0.12) for low ABI and 2.48 (95% CI [1.41; 4.36] p=0.002) for very low ABI compared to pts with normal ABI. Including ABI in the prediction model significantly increased the C-statistic (from 0.80 to 0.82, p=0.03).

Conclusion: ABI is a strong and independent long term predictor factor of all-cause death in CAD. To better identify pts at very high risk, systematic ABI assessment should be promoted.

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The relationship between vascular dysfunction and angina with angiographically normal coronary arteries

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Background: Angina with angiographically normal coronary arteries (NCA) still has controversial pathophysiological mechanisms. Experimental studies have shown that coronary blood flow varies with aortic (Ao) stiffness, but clinical data are poor.

Objective: To assess Ao vascular function by different echocardiographic techniques in patients (pts) with angina and NCA vs asymptomatic pts.

Methods: Study group consisted of 15 pts with angina and NCA (mean age 60,9±5,3 yrs) and 15 age and gender- matched control subjects (normal and hypertensives pts) .

Ascending Ao diameters – derived stiffness indices were: Ao strain, Ao distensibility (Ao dis), Ao stiffness index (Ao SI). Ao function was evaluated also by measuring tissue Doppler (TdI) systolic (SW), early and late diastolic(EW, AW) velocities of the anterior Ao wall. Total afterload was defined by the effective arterial elastance (Ea=0.9XSBP/SV, SV=stroke volume). Systemic vascular resistance index (SVRI)= mean arterial pressure/cardiac index. Total arterial compliance (Ca)=SV/pulse pressure.

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